=> d his

(FILE 'HOME' ENTERED AT 14:43:24 ON 06 JUN 2006)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, ANTE, AQUALINE, AQUASCI, BIOENG, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CAPLUS, CEABA-VTB, CIN, CONFSCI, CROPB, CROPU, DDFB, DDFU, DGENE, DISSABS, DRUGB, DRUGMONOG2, DRUGU, EMBAL, EMBASE, ...' ENTERED AT 14:43:35 ON 06 JUN 2006

SEA GLUCOSE TRANSPORTER

153	FILE ADISCTI
10	FILE ADISINSIGHT
6	FILE ADISNEWS
411	FILE AGRICOLA
15	FILE ANABSTR
1	FILE ANTE
33	FILE AQUASCI
109	FILE BIOENG
7222	FILE BIOSIS
126	FILE BIOTECHABS
126	FILE BIOTECHDS
2775	FILE BIOTECHNO
746	FILE CABA
6881	FILE CAPLUS
8	FILE CEABA-VTB
9	FILE CIN
155	FILE CONFSCI
1	FILE CROPU
1	FILE DDFB
361	FILE DDFU
1591	FILE DGENE
278	FILE DISSABS
1	FILE DRUGB
447	FILE DRUGU
82	FILE EMBAL
7167	FILE EMBASE
2977	FILE ESBIOBASE
31	FILE FROSTI
26	FILE FSTA
2298	FILE GENBANK
1	FILE HEALSAFE
148	FILE IFIPAT
16	FILE IMSDRUGNEWS
10	FILE IMSRESEARCH
818	FILE JICST-EPLUS
2	FILE KOSMET
1483	FILE LIFESCI
6748	FILE MEDLINE
10	FILE NTIS
7	FILE OCEAN
2351	FILE PASCAL
15	FILE PHAR
5	FILE PHARMAML
14	FILE PHIN
52	FILE PROMT
127	FILE PROUSDDR
1	FILE RDISCLOSURE
7091	FILE SCISEARCH
2389	FILE TOXCENTER
1755	FILE USPATFULL
154	FILE USPAT2
9	FILE VETU

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             187   FILE WPIDS
               7   FILE WPIFV
             187   FILE WPINDEX
L1              QUE GLUCOSE TRANSPORTER
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     FILE 'BIOSIS, EMBASE, SCISEARCH, CAPLUS, MEDLINE, ESBIOBASE, BIOTECHNO,
     TOXCENTER, PASCAL' ENTERED AT 14:44:34 ON 06 JUN 2006
L2         15132 S L1 AND (GLUT4 OR GLUT-4 OR GLUTIV OR GLUT-IV)
L3             0 S L2 AND GLUT4V85M
L4          3784 S L2 AND HUMAN
L5            15 S L4 AND VALINE
L6             6 DUP REM L5 (9 DUPLICATES REMOVED)
L7             0 S L4 AND (VARIANTS AND MUTANTS)
L8            12 S L4 AND (VARIANT AND MUTANT)
L9           244 S L4 AND (VARIANT OR MUTANT)
L10          101 DUP REM L9 (143 DUPLICATES REMOVED)
L11            3 S L10 AND (VALINE OR METHIONINE)
L12            3 DUP REM L11 (0 DUPLICATES REMOVED)
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=> d l12 ibib ab 1-3

L12 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2004:218475 CAPLUS
DOCUMENT NUMBER: 140:250812
TITLE: Use of erg4 **mutants** of Saccharomyces cerevisiae as hosts for the expression of of genes for mammalian **glucose transporters**
INVENTOR(S): Mueller, Guenter; Dlugai, Silke; Voss, Doerthe; Boles, Eckhard
PATENT ASSIGNEE(S): Aventis Pharma Deutschland G.m.b.H., Germany
SOURCE: Ger. Offen., 20 pp.
CODEN: GWXXBX
DOCUMENT TYPE: Patent
LANGUAGE: German
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
DE 10242763	A1	20040318	DE 2002-10242763	20020914
CA 2498636	AA	20040401	CA 2003-2498636	20030904
WO 2004026907	A2	20040401	WO 2003-EP9812	20030904
WO 2004026907	A3	20041111		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
AU 2003264257	A1	20040408	AU 2003-264257	20030904
EP 1539958	A2	20050615	EP 2003-797264	20030904
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK				
BR 2003014115	A	20050712	BR 2003-14115	20030904
US 2005074845	A1	20050407	US 2003-659234	20030910
ZA 2005001871	A	20050907	ZA 2005-1871	20050304
NO 2005001795	A	20050608	NO 2005-1795	20050412

PRIORITY APPLN. INFO.:			
DE 2002-10242763	A	20020914	
US 2003-455340P	P	20030317	
WO 2003-EP9812	W	20030904	

AB As well as the invention refers to yeast trunks, in those a **human** **GLUT4**-Transporter or a **human** GLUT1-Transporter functionally to the expression to be brought can to certain **GLUT4** -Transportproteine, which can be particularly simply functionally expressed in yeast trunks. Saccharomyces cerevisiae strains defective in glucose transport due to mutation in the FGY1 and ERG4 genes can be used as expression hosts for mammalian GLUT1 and **GLUT4** transporter genes. The appearance of **GLUT4** activity is improved by a point mutation leading to a substitution of 85-**valine** by **methionine**. Use of combinations of alleles of the FGY1, ERG4 and ERG5 genes to improve the level of GLUT1 or **GLUT4**-mediated glucose transport is demonstrated.
REFERENCE COUNT: 1 THERE ARE 1 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L12 ANSWER 2 OF 3 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 1992:96110 BIOSIS
DOCUMENT NUMBER: PREV199293052660; BA93:52660

TITLE: MOLECULAR SCANNING OF INSULIN-RESPONSIVE **GLUCOSE TRANSPORTER GLUT4** GENE IN NIDDM SUBJECTS.
AUTHOR(S): CHOI W-H [Reprint author]; O'RAHILLY S; BUSE J B; REES A; MORGAN R; FLIER J S; MOLLER D E
CORPORATE SOURCE: BETH ISRAEL HOSP, SL 436, 330 BROOKLINE AVE, BOSTON, MASS 02215, USA
SOURCE: Diabetes, (1991) Vol. 40, No. 12, pp. 1712-1718.
CODEN: DIAEAZ. ISSN: 0012-1797.
DOCUMENT TYPE: Article
FILE SEGMENT: BA
LANGUAGE: ENGLISH
ENTRY DATE: Entered STN: 12 Feb 1992
Last Updated on STN: 13 Feb 1992

AB We investigated the prevalence of mutations in the gene encoding the major insulin-responsive facilitative **glucose transporter** (**GLUT4**) in patients with non-insulin-dependent diabetes mellitus (NIDDM). All 11 exons of the **GLUT4** gene from 30 British white subjects with NIDDM were amplified using the polymerase chain reaction and screened for nucleotide sequence variation using the single-stranded conformation polymorphism (SSCP) method. No variation between the study subjects was detected in exons 1-3, 4b-8, and 10. **Variant** SSCP patterns were detected in exons 4a and 9. SSCP variation in exon 4a was revealed by direct nucleotide sequencing to be due to a common silent polymorphism (AAC→AAT at Asn130). One NIDDM patient demonstrated a **variant** SSCP pattern in exon 9. This was caused by a point mutation (GTC→ATC) at codon 383, which leads to the conservative substitution of isoleucine for **valine** in the putative fifth extracellular loop of the transporter. Allele-specific oligonucleotide hybridization was used to examine the frequency of this mutation in 240 Welsh white subjects (160 with NIDDM and 80 controls). The Val→Ile383 mutation was found in the heterozygous state in two diabetic subjects and no control subjects. We conclude that mutations of the **GLUT4** coding sequence are very uncommon in this population of subjects with typical NIDDM. Determining whether the IIe383 **GLUT4 variant** present in 3 diabetic subjects contributes in any way to their disease will require further study.

L12 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 1991:672182 CAPLUS
DOCUMENT NUMBER: 115:272182
TITLE: Analysis of the gene sequences of the insulin receptor and the insulin-sensitive **glucose transporter** (**GLUT-4**) in patients with common-type non-insulin-dependent diabetes mellitus
AUTHOR(S): Kusari, J.; Verma, U. S.; Buse, J. B.; Henry, R. R.; Olefsky, J. M.
CORPORATE SOURCE: Dep. Med., Univ. California, San Diego, La Jolla, CA, 92093, USA
SOURCE: Journal of Clinical Investigation (1991), 88(4), 1323-30
CODEN: JCINAO; ISSN: 0021-9738
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Insulin resistance is a common feature of non-insulin-dependent diabetes mellitus (NIDDM) and "diabetes susceptibility genes" may be involved in this abnormality. Two potential candidate genes are the insulin receptor (IR) and the insulin-sensitive **glucose transporter** (**GLUT-4**). To elucidate whether structural defects in the IR and/or **GLUT-4** could be a primary cause of insulin resistance in NIDDM, the entire coding region of the **GLUT-4** gene from DNA of 6 NIDDM patients was sequenced. Since binding properties of the IRs from NIDDM subjects are normal, the sequence of

exons 16-22 (encoding the entire cytoplasmic domain of the IR) of the IR gene from the same six patients was also analyzed. When compared with the normal IR sequence, no difference was found in the predicted amino acid sequence of the IR cytoplasmic domain derived from the NIDDM patients. Sequence anal. of the **GLUT-4** gene revealed that one patient was heterozygous for a mutation in which isoleucine (ATC) was substituted for **valine** (GTC) at position 383. Consequently, the **GLUT-4** sequence at position 383 was determined in 24 addnl. NIDDM patients and 30 nondiabetic controls and all showed only the normal sequence. Thus, the insulin resistance seen in the great majority of subjects with the common form of NIDDM is not due to genetic variation in the coding sequence of the IR β subunit, nor to any single mutation in the **GLUT-4** gene. Possibly, a subpopulation of NIDDM patients exists displaying variation in the **GLUT-4** gene.